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Terms	Documents
11 and 12 and 15	11

US Patents Full-Text Database
 US Pre-Grant Publication Full-Text Database
 JPO Abstracts Database
 EPO Abstracts Database
 Derwent World Patents Index
 IBM Technical Disclosure Bulletins

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Search History

DATE: Thursday, April 18, 2002 [Printable Copy](#) [Create Case](#)

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side by side		

<u>Hit Count</u>	<u>Set Name</u>	
result set		

DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=YES; OP=OR

<u>L7</u>	11 and 12 and 15	11	<u>L7</u>
<u>L6</u>	L5 and osteoartheritic with knee	0	<u>L6</u>
<u>L5</u>	L2 and liquid\$ or amorphous and artheriti\$	11	<u>L5</u>
<u>L4</u>	L2 and biabsorb and carrier	0	<u>L4</u>
<u>L3</u>	L2 and liquid\$ or amorphous	178642	<u>L3</u>
<u>L2</u>	L1 and tissue with repair	15	<u>L2</u>
<u>L1</u>	polyhydroxyalkanoate	543	<u>L1</u>

END OF SEARCH HISTORY

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*R. S. M.
1/14/99*
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Search Results - Record(s) 11 through 15 of 15 returned.

 11. Document ID: US 5863531 A

L2: Entry 11 of 15

File: USPT

Jan 26, 1999

US-PAT-NO: 5863531

DOCUMENT-IDENTIFIER: US 5863531 A

TITLE: In vitro preparation of tubular tissue structures by stromal cell culture on a three-dimensional framework

DATE-ISSUED: January 26, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Naughton; Gail K.	Del Mar	CA		
Naughton; Brian A.	El Cajon	CA		

US-CL-CURRENT: 424/93.7; 424/423, 435/174, 435/180, 435/182, 435/395, 435/398[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#) | [Claims](#) | [RWN](#) | [Drawn Desc](#) | [Image](#) 12. Document ID: US 5842477 A

L2: Entry 12 of 15

File: USPT

Dec 1, 1998

US-PAT-NO: 5842477

DOCUMENT-IDENTIFIER: US 5842477 A

TITLE: Method for repairing cartilage

DATE-ISSUED: December 1, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Naughton; Gail K.	Del Mar	CA		
Willoughby; Jane	Solana Beach	CA		

US-CL-CURRENT: 128/898; 623/902[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#) | [Claims](#) | [RWN](#) | [Drawn Desc](#) | [Image](#) 13. Document ID: WO 200119422 A1, AU 200112523 A

L2: Entry 13 of 15

File: DWPI

Mar 22, 2001

DERWENT-ACC-NO: 2001-374211

DERWENT-WEEK: 200139

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TITLE: Compositions comprising polyhydroxyalkanoate polymers, for repair of soft

tissue, augmentation, and as viscosupplements, e.g. in osteoarthritic knees

INVENTOR: MARTIN, D P; WILLIAMS, S F

PRIORITY-DATA: 1999US-153810P (September 14, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200119422 A1	March 22, 2001	E	024	A61L027/18
AU 200112523 A	April 17, 2001		000	A61L027/18

INT-CL (IPC): A61 L 27/18

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#) [KWD](#) [Drawn Desc](#) [Image](#)

14. Document ID: EP 1163019 A1, WO 200056376 A1, AU 200040277 A

L2: Entry 14 of 15

File: DWPI

Dec 19, 2001

DERWENT-ACC-NO: 2000-579476

DERWENT-WEEK: 200206

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TITLE: Biodegradable polyhydroxyalkanoate polymer composition, useful for medical devices e.g. sutures, tissue repair devices, bone grafts and wound dressings, have controlled degradation rates of less than two years

INVENTOR: MARTIN, D P; SKRALY, F ; WILLIAMS, S F

PRIORITY-DATA: 1999US-142238P (July 2, 1999), 1999US-126180P (March 25, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1163019 A1	December 19, 2001	E	000	A61L031/14
WO 200056376 A1	September 28, 2000	E	069	A61L031/14
AU 200040277 A	October 9, 2000		000	A61L031/14

INT-CL (IPC): A61 L 17/12; A61 L 27/18; A61 L 27/58; A61 L 31/06; A61 L 31/14

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#) [KWD](#) [Drawn Desc](#) [Image](#)

15. Document ID: EP 1159015 A1, WO 200051662 A1, AU 200037228 A

L2: Entry 15 of 15

File: DWPI

Dec 5, 2001

DERWENT-ACC-NO: 2000-579231

DERWENT-WEEK: 200203

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TITLE: Biocompatible, bioabsorbable polymers with mechanical properties that provide a better match with those of tissue structures

INVENTOR: WILLIAMS, S F

PRIORITY-DATA: 1999US-122827P (March 4, 1999)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
EP 1159015 A1	December 5, 2001	E	000	A61L031/06
WO 200051662 A1	September 8, 2000	E	027	A61L031/06
AU 200037228 A	September 21, 2000		000	A61L031/06

INT-CL (IPC): A61 L 31/06[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#)[KMC](#) | [Drawn Desc](#) | [Image](#)[Generate Collection](#)[Print](#)

Terms	Documents
L1 and tissue with repair	15

[Display Format:](#) [Change Format](#)[Previous Page](#) [Next Page](#)

WEST[Generate Collection](#)[Print](#)**Search Results - Record(s) 1 through 10 of 15 returned.** 1. Document ID: US 20020045567 A1

L2: Entry 1 of 15

File: PGPB

Apr 18, 2002

PGPUB-DOCUMENT-NUMBER: 20020045567
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020045567 A1

TITLE: SYNTHETIC PROTEINS FOR IN VIVO DRUG DELIVERY AND TISSUE AUGMENTATION

PUBLICATION-DATE: April 18, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
CAPPELLO, JOSEPH	SAN DIEGO	CA	US	
STEDRONSKY, ERWIN R.	SAN DIEGO	CA	US	

US-CL-CURRENT: 514/2; 435/69.1, 514/17, 530/329, 530/330, 530/331, 530/332[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#)[KMC](#) | [Drawn Desc](#) | [Image](#) 2. Document ID: US 20020034757 A1

L2: Entry 2 of 15

File: PGPB

Mar 21, 2002

PGPUB-DOCUMENT-NUMBER: 20020034757
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020034757 A1

TITLE: Single-molecule selection methods and compositions therefrom

PUBLICATION-DATE: March 21, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Cubicciotti, Roger S.	Montclair	NJ	US	

US-CL-CURRENT: 435/6; 435/91.2, 536/22.1, 536/23.1, 536/24.3[Full](#) | [Title](#) | [Citation](#) | [Front](#) | [Review](#) | [Classification](#) | [Date](#) | [Reference](#) | [Sequences](#) | [Attachments](#)[KMC](#) | [Drawn Desc](#) | [Image](#) 3. Document ID: US 20010044651 A1

L2: Entry 3 of 15

File: PGPB

Nov 22, 2001

PGPUB-DOCUMENT-NUMBER: 20010044651
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20010044651 A1

TITLE: Expandable stent with sliding and locking radial elements

PUBLICATION-DATE: November 22, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Steinke, Thomas A.	San Diego	CA	US	
Koenig, Donald H.	San Diego	CA	US	

US-CL-CURRENT: 623/1.16; 623/1.17

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)

[HTMLC](#) [Drawn Desc](#) [Image](#)

4. Document ID: US 20010044413 A1

L2: Entry 4 of 15

File: PGPB

Nov 22, 2001

PGPUB-DOCUMENT-NUMBER: 20010044413

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20010044413 A1

TITLE: In situ bioreactors and methods of use thereof

PUBLICATION-DATE: November 22, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Pierce, Glenn	Rancho Santa Fe	CA	US	
Chandler, Lois Ann	Encinitas	CA	US	

US-CL-CURRENT: 514/44

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)

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5. Document ID: US 6368343 B1

L2: Entry 5 of 15

File: USPT

Apr 9, 2002

US-PAT-NO: 6368343

DOCUMENT-IDENTIFIER: US 6368343 B1

TITLE: Method of using ultrasonic vibration to secure body tissue

DATE-ISSUED: April 9, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bonutti; Peter M.	Effingham	IL	62401	
Cremens; Matthew J.	Effingham	IL		
Ruholl; Kevin	Teutopolis	IL		

US-CL-CURRENT: 606/232; 606/144

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)

[HTMLC](#) [Drawn Desc](#) [Image](#)

6. Document ID: US 6291240 B1

L2: Entry 6 of 15

File: USPT

Sep 18, 2001

US-PAT-NO: 6291240

DOCUMENT-IDENTIFIER: US 6291240 B1

TITLE: Cells or tissues with increased protein factors and methods of making and using same

DATE-ISSUED: September 18, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Mansbridge; Jonathan N.	La Jolla	CA		
Liu; Kang	San Diego	CA		

US-CL-CURRENT: 435/395; 435/1.3, 435/325, 435/347, 435/373, 435/402, 435/455

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	KMM	Drawn Desc	Image
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 7. Document ID: US 6287765 B1

L2: Entry 7 of 15

File: USPT

Sep 11, 2001

US-PAT-NO: 6287765

DOCUMENT-IDENTIFIER: US 6287765 B1

TITLE: Methods for detecting and identifying single molecules

DATE-ISSUED: September 11, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Cubicciotti; Roger S.	Montclair	NJ		

US-CL-CURRENT: 435/6; 435/91.2, 536/22.1, 536/23.1, 536/24.3, 536/24.5

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	KMM	Drawn Desc	Image
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 8. Document ID: US 6140039 A

L2: Entry 8 of 15

File: USPT

Oct 31, 2000

US-PAT-NO: 6140039

DOCUMENT-IDENTIFIER: US 6140039 A

TITLE: Three-dimensional filamentous tissue having tendon or ligament function

DATE-ISSUED: October 31, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Naughton; Gail K.	Del Mar	CA		
Naughton; Brian A.	El Cajon	CA		

US-CL-CURRENT: 435/1.1; 424/423, 424/93.7, 435/177, 435/178, 435/179, 435/180, 435/395,
435/398, 435/399, 435/402

[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)
[HTML](#) [Draw Desc](#) [Image](#)
 9. Document ID: US 6127166 A

L2: Entry 9 of 15

File: USPT

Oct 3, 2000

US-PAT-NO: 6127166

DOCUMENT-IDENTIFIER: US 6127166 A

TITLE: Molluscan ligament polypeptides and genes encoding them

DATE-ISSUED: October 3, 2000

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Bayley; Hagan	College Station	TX	77845	
Cao; Qiuping	Shrewsbury	MA	01545	
Wang; Yunjuan	Bryan	TX	77801	

US-CL-CURRENT: 435/252.3; 435/320.1, 435/325, 435/69.1, 536/23.1, 536/23.5
[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)
[HTML](#) [Draw Desc](#) [Image](#)
 10. Document ID: US 5919702 A

L2: Entry 10 of 15

File: USPT

Jul 6, 1999

US-PAT-NO: 5919702

DOCUMENT-IDENTIFIER: US 5919702 A

TITLE: Production of cartilage tissue using cells isolated from Wharton's jelly

DATE-ISSUED: July 6, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Purchio; Anthony F.	La Jolla	CA		
Naughton; Brian A.	El Cajon	CA		
San Roman; Julia	San Diego	CA		

US-CL-CURRENT: 435/378; 424/93.1, 435/325, 435/366, 435/377
[Full](#) [Title](#) [Citation](#) [Front](#) [Review](#) [Classification](#) [Date](#) [Reference](#) [Sequences](#) [Attachments](#)
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Terms	Documents
L1 and tissue with repair	15

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NEWS 2 Jan 25 BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3 Jan 25 Searching with the P indicator for Preparations
NEWS 4 Jan 29 FSTA has been reloaded and moves to weekly updates
NEWS 5 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 6 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 7 Mar 08 Gene Names now available in BIOSIS
NEWS 8 Mar 22 TOXLIT no longer available
NEWS 9 Mar 22 TRCTHERMO no longer available
NEWS 10 Mar 28 US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 11 Mar 28 LIPINSKI/CALC added for property searching in REGISTRY
NEWS 12 Apr 02 PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 13 Apr 08 "Ask CAS" for self-help around the clock
NEWS 14 Apr 09 BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 15 Apr 09 ZDB will be removed from STN

NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d,
CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002

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FILE 'HOME' ENTERED AT 09:13:13 ON 18 APR 2002

=> file medline, caplus, scisearch, biosis, embase
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FULL ESTIMATED COST 1.05 1.05

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FILE 'CAPLUS' ENTERED AT 09:16:02 ON 18 APR 2002
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=> polyhydroxyalkanoate
POLYHYDROXYALKANOATE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s polyhydroxyalkanate
L1 3 POLYHYDROXYALKANATE

=> s polyhydroxyalkanate and bioabsorb
=> s polyhydroxyalkanate(n)bioabsorb
L2 0 POLYHYDROXYALKANATE(N) BIOABSORB

=> s polyhydroxyalkanate(n) repair
L3 0 POLYHYDROXYALKANATE(N) REPAIR

=> s polyhydroxyalkanate(n) repair(w) tissue
L4 0 POLYHYDROXYALKANATE(N) REPAIR(W) TISSUE

=> s l1 and tissue(w) repair
L5 0 L1 AND TISSUE(W) REPAIR

=> s l1 and liquid(w) carrier
3 FILES SEARCHED...
L6 0 L1 AND LIQUID(W) CARRIER

=> s l1 and liquid(w) carrier and biocompatible
4 FILES SEARCHED...
L7 0 L1 AND LIQUID(W) CARRIER AND BIOCOMPATIBLE

=> s l1 and liquid(w) carrier and biocompatible and compounds or anti(w) microbial
2 FILES SEARCHED...
L8 8600 L1 AND LIQUID(W) CARRIER AND BIOCOMPATIBLE AND COMPOUNDS OR
ANTI(W) MICROBIAL

=> s l8 and tissue(w) repair
L9 3 L8 AND TISSUE(W) REPAIR

=> s l8 and anesthetic or adjuvants or anti(w)inflammator or surfactants or steroid
or lipid or enzyme or antibodie or hormone
=> s l8 and anesthetic or adjuvants or anti(w)inflammator or surfactants or steroid
or lipid or enzyme or antibodie or hormone
4 FILES SEARCHED...

L10 6975460 L8 AND ANESTHETIC OR ADJUVANTS OR ANTI(W) INFLAMMATOR OR SURFACTANTS OR STEROID OR LIPID OR ENZYME OR ANTIBODIE OR HORMONE

=> s 11 and anesthetic or adjuvants or anti(w)inflammator or surfactants or steroid or lipid or enzyme or antibodie or hormone
3 FILES SEARCHED...

L11 6975467 LL AND ANESTHETIC OR ADJUVANTS OR ANTI(W) INFLAMMATOR OR SURFACTANTS OR STEROID OR LIPID OR ENZYME OR ANTIBODIE OR HORMONE

=> s 111 and osteoarthritic(w) knee

L12 37 L11 AND OSTEOARTHRITIC(W) KNEE

=> s 112 and amorphous

L13 0 L12 AND AMORPHOUS

=> s 112 and treatment

L14 9 L12 AND TREATMENT

=> d 114 1-9 ibib abs

L14 ANSWER 1 OF 9 MEDLINE

ACCESSION NUMBER: 96016512 MEDLINE

DOCUMENT NUMBER: 96016512 PubMed ID: 7586773

TITLE: The levels of collagenase, tissue inhibitor of metalloproteinases-1 (TIMP-1), collagenase approximately TIMP-1 complexes and glycosaminoglycan (GAG) in sequential samples of synovial fluid aspirated from patients with osteoarthritis.

AUTHOR: Cawston T E; Curry V; Ramsey S; Clark I M; Kyle V A; Adebajo A; Silverman B; Daymond T; Hazleman B L

CORPORATE SOURCE: Rheumatology Research Unit, Addenbrooke's Hospital, Cambridge, UK.

SOURCE: CLINICAL AND EXPERIMENTAL RHEUMATOLOGY, (1995 Jul-Aug) 13 (4) 431-7.

Journal code: DFA; 8308521. ISSN: 0392-856X.

PUB. COUNTRY: Italy

(CLINICAL TRIAL)

Journal; Article; (JOURNAL ARTICLE)

(RANDOMIZED CONTROLLED TRIAL)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199512

ENTRY DATE: Entered STN: 19960124

Last Updated on STN: 19980206

Entered Medline: 19951212

AB OBJECTIVE. Collagen turnover in connective tissues is thought to be controlled by the balance between the levels of interstitial collagenase and tissue inhibitor of metalloproteinases (TIMP-1). The aim of this study was to measure the level of total collagenase (MMP-1), TIMP-1, collagenase approximately TIMP-1 complex and glycosaminoglycan (GAG) in sequential samples of **osteoarthritic knee** synovial fluid from well documented patients to determine if these parameters changed with time and correlated with clinical indices. METHODS. Twenty-one patients were recruited and randomly allocated to receive tiaprofenic acid, indomethacin or naproxen. Total collagenase, TIMP-1, collagenase approximately TIMP-1 complex and GAG were measured in 80 osteoarthritic synovial fluids taken over a period of six months. RESULTS. The majority of fluids contained a molar excess of TIMP-1 over collagenase, although in seven fluids collagenase was present in excess; six of these samples were from a single patient. GAG levels were relatively unchanged over the six months studied. CONCLUSION. The levels of collagenase and TIMP-1 varied between patients and over time in individual patients. No collagenase approximately TIMP-1 complex was found in any fluid. There was no significant difference in the median levels of collagenase, TIMP-1 or GAG

in the different **treatment** groups. High levels of collagenase were found in one patient with a crystal related disease. These immunoassays give valuable information on the levels of collagenase and TIMP-1 in individual patients with time and may help to determine the mechanisms controlling the turnover of cartilage collagen in different arthritides.

L14 ANSWER 2 OF 9 MEDLINE
ACCESSION NUMBER: 89061173 MEDLINE
DOCUMENT NUMBER: 89061173 PubMed ID: 3196082
TITLE: Activation of neutral metalloprotease in human **osteoarthritic knee** cartilage: evidence for degradation in the core protein of sulphated proteoglycan.
AUTHOR: Martel-Pelletier J; Pelletier J P; Malemud C J
CORPORATE SOURCE: Unite des Maladies Rhumatismales, Hopital Notre-Dame, University of Montreal, Quebec, Canada.
CONTRACT NUMBER: AG-02205 (NIA)
SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (1988 Oct) 47 (10) 801-8.
JOURNAL code: 62W; 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198901
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 20000303
Entered Medline: 19890112

AB The neutral, metal dependent, proteoglycan degrading **enzymes** (NMPEs) in human **osteoarthritic knee** cartilage homogenates were activated by p-aminophenylmercuric acetate (APMA). The resultant effect on the structure of newly synthesised and already existing sulphated proteoglycan was measured. Newly synthesised and already existing proteoglycan aggregated to hyaluronic acid was reduced (p less than 0.01, p less than 0.05 respectively) when measured by chromatography on Sepharose CL-2B eluted with associative buffer. The APMA activated **enzyme** affected both the newly synthesised and already existing proteoglycan aggregate similarly ($r = 0.79$, p less than 0.001). **Treatment** of cartilage homogenates with APMA and 1,10-phenanthroline (10 mM) showed that the amount of aggregated proteoglycan was at the control level. The hydrodynamic size of the proteoglycan monomer (A1D1) was also reduced by **treatment** of cartilage homogenates with APMA. Reaggregation experiments with fraction A1D1 and exogenous hyaluronic acid and link protein showed a similar defect in forming proteoglycan aggregates. These data showed that activation of the NMPEs altered the structure of proteoglycan in two ways. The most consistent change was a reduction in the ability of proteoglycan to form aggregates with hyaluronic acid. This was likely to have occurred via a cleavage of the core protein in or around the hyaluronic acid binding globular domain. A second alteration probably includes a limited proteolytic cleavage in the remainder of the core protein.

L14 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:527428 CAPLUS
DOCUMENT NUMBER: 129:145637
TITLE: Human apoptosis-associated protein-encoding DNA is similar to p53 response mouse gene E124
INVENTOR(S): Hillman, Jennifer L.; Goli, Surya K.
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9832854	A1	19980730	WO 1998-US1421	19980126
W: AT, AU, BR, CA, CH, CN, DE, DK, ES, FI, GB, IL, JP, KR, MX, NO, NZ, RU, SE, SG, US, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 5858715	A	19990112	US 1997-790572	19970129
AU 9860412	A1	19980818	AU 1998-60412	19980126
EP 1012272	A1	20000628	EP 1998-903716	19980126
R: BE, DE, ES, FR, GB, IT, NL				
JP 2001509018	T2	20010710	JP 1998-532187	19980126
US 5955429	A	19990921	US 1998-213398	19981215
PRIORITY APPLN. INFO.:			US 1997-790572	A2 19970129
			WO 1998-US1421	W 19980126

AB The present invention provides a novel human apoptosis-assocd. protein (NHAAP) and polynucleotides which identify and encode NHAAP. Nucleic acids encoding human NHAAP were first identified in Incyte clone 723748 from an **osteoarthritic knee** joint cDNA library through a computer-generated search for amino acid sequence alignments; a consensus sequence was derived from overlapping and/or extended nucleic acid sequences. NHAAP is 340 amino acids in length and has chem. and structural homol. with mouse E124. Northern anal. shows the expression of this sequence in various libraries, at least 52% of which are derived from immortalized or cancerous cells and at least 20% of which are of fetal origin. The invention also provides genetically engineered expression vectors and host cells comprising the nucleic acid sequences encoding NHAAP and a method for producing NHAAP. The invention also provides for agonists, **antibodies**, or antagonists specifically binding NHAAP, and their use, in the prevention and **treatment** of diseases assocd. with expression of NHAAP. Addnl., the invention provides for the use of antisense mols. to polynucleotides encoding NHAAP for the **treatment** of diseases assocd. with the expression of NHAAP. The invention also provides diagnostic assays which utilize the polynucleotide, or fragments or the complement thereof, and **antibodies** specifically binding NHAAP.

L14 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1989:5722 CAPLUS

DOCUMENT NUMBER: 110:5722

TITLE: Activation of neutral metalloprotease in human **osteoarthritic knee** cartilage: evidence for degradation in the core protein of sulfated proteoglycan

AUTHOR(S): Martel-Pelletier, Johanne; Pelletier, Jean Pierre; Malemud, Charles J.

CORPORATE SOURCE: Res. Cent., Hop. Notre-Dame, Montreal, PQ, Can.

SOURCE: Ann. Rheum. Dis. (1988), 47(10), 801-8

CODEN: ARDIAO; ISSN: 0003-4967

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The neutral, metal-dependent, proteoglycan-degrading **enzymes** (NMPEs) in human **osteoarthritic knee** cartilage homogenates were activated by p-aminophenylmercuric acetate (APMA). The resultant effect on the structure of newly synthesized and already existing sulfated proteoglycan was measured. Newly synthesized and already existing proteoglycan aggregated to hyaluronic acid was reduced

when measured by chromatog. on Sepharose CL-2B eluted with associative buffer. The APMA activated **enzyme** affected both the newly synthesized and already existing proteoglycan aggregate similarly. **Treatment** of cartilage homogenates with APMA and 1,10-phenanthroline (10 mM) showed that the amt. of aggregated proteoglycan was at the control level. The hydrodynamic size of the proteoglycan monomer (A1D1) was also reduced by **treatment** of cartilage homogenates with APMA. Reaggregation expts. with fraction A1D1 and exogenous hyaluronic acid and link protein showed a similar defect in forming proteoglycan aggregates. Thus, activation of the NMPEs altered the structure of proteoglycan in 2 ways. The most consistent change was a redn. in the ability of proteoglycan to form aggregates with hyaluronic acid. This was likely have occurred via a cleavage of the core protein in or around the hyaluronic acid binding globular domain. A second alteration probably includes a limited proteolytic cleavage in the remainder of the core protein.

L14 ANSWER 5 OF 9 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 1999:519203 SCISEARCH
THE GENUINE ARTICLE: 210WY
TITLE: Degenerative disease of the knee joint: efficacy and contribution of local **treatment**
AUTHOR: Ayrat X (Reprint)
CORPORATE SOURCE: HOP COCHIN, SERV RHUMATOL B, 27 RUE FAUBOURG ST JACQUES, F-75014 PARIS, FRANCE (Reprint)
COUNTRY OF AUTHOR: FRANCE
SOURCE: PRESSE MEDICALE, (19 JUN 1999) Vol. 28, No. 22, pp. 1195-1200.
Publisher: MASSON EDITEUR, 120 BLVD SAINT-GERMAIN, 75280 PARIS 06, FRANCE.
ISSN: 0755-4982.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: French
REFERENCE COUNT: 29

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Indications: To relieve pain in patients with knee osteoarthritis, local **treatments** can be effective both for episodes of acute congestion, characterized by inflammatory pain, intraarticular effusion and risk of acute chondrolysis, and for slowly progressive disease (with a characteristic lack of effusion).

Acute congestion: Local care is essential. Relief can be achieved by draining the effusion, associated with corticosteroid injections which may be repeated and followed by a 24 h rest. In case of failure or rapid development of chondrolysis, joint ravage (1 liter saline solution - two 2-mm needles) followed by cortico-steroid infiltration is indicated. Weight bearing should be avoided for 6 weeks (cane) until the effusion has been absorbed. In case of radiological evidence of chondrocalcinosis and chronic serous or bloody effusion, yttrium 90 synoviorthesis may be proposed as an alternative.

Slowly progressive disease: In patients with no effusion who continue to suffer despite physical and medical **treatment**, intraarticular injections of hyaluronic acid can be helpful. They are particularly effective in case of moderate disease. Hyaluronic acid is an interesting alternative to non-steroidal antiinflammatory drugs and is especially indicated after a rapidly progressive period of chondrolysis.

L14 ANSWER 6 OF 9 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 95:546626 SCISEARCH
THE GENUINE ARTICLE: RN184
TITLE: THE LEVELS OF COLLAGENASE, TISSUE INHIBITOR OF METALLOPROTEINASES-1 (TIMP-1), COLLAGENASE-TIMP-1

COMPLEXES AND GLYCOSAMINOGLYCAN (GAG) IN SEQUENTIAL SAMPLES OF SYNOVIAL-FLUID ASPIRATED FROM PATIENTS WITH OSTEOARTHRITIS

AUTHOR: CAWSTON T E (Reprint); CURRY V; RAMSEY S; CLARK I M; KYLE V A; ADEBAJO A; SILVERMAN B; DAYMOND T; HAZLEMAN B L

CORPORATE SOURCE: ADDENBROOKES HOSP, RHEUMATOL RES UNIT, HILLS RD, CAMBRIDGE CB2 2QQ, ENGLAND (Reprint); ROYAL INFIRM, DEPT RHEUMATOL, SUNDERLAND, DURHAM, ENGLAND; FRENCHAY HOSP, DEPT MED, BRISTOL BS16 1LE, AVON, ENGLAND

COUNTRY OF AUTHOR: ENGLAND

SOURCE: CLINICAL AND EXPERIMENTAL RHEUMATOLOGY, (JUL/AUG 1995) Vol. 13, No. 4, pp. 431-437.

ISSN: 0392-856X.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE; CLIN

LANGUAGE: ENGLISH

REFERENCE COUNT: 34

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective. Collagen turnover in connective tissues is thought to be controlled by the balance between the levels of interstitial collagenase and tissue inhibitor of metalloproteinases (TIMP-1). The aim of this study was to measure the level of total collagenase (MMP-1), TIMP-1, collagenase similar to TIMP-1 complex and glycosaminoglycan (GAG) in sequential samples of **osteoarthritic knee** synovial fluid from well documented patients to determine if these parameters changed with time and correlated with clinical indices.

Methods. Twenty-one patients were recruited and randomly allocated to receive tiaprofenic acid, indomethacin or naproxen. Total collagenase, TIMP-1, collagenase similar to TIMP-1 complex and GAG were measured in 80 osteoarthritic synovial fluids taken over a period of six months.

Results. The majority of fluids contained a molar excess of TIMP-1 over collagenase, although in seven fluids collagenase was present in excess; six of these samples were from a single patient. GAG levels were relatively unchanged over the six months studied.

Conclusion. The levels of collagenase and TIMP-1 varied between patients and over time in individual patients. No collagenase similar to TIMP-1 complex was found in any fluid. There was no significant difference in the median levels of collagenase, TIMP-1 or GAG in the different **treatment** groups. High levels of collagenase were found in one patient with a crystal related disease. These immunoassays give valuable information on the levels of collagenase and TIMP-1 in individual patients with time and may help to determine the mechanisms controlling the turnover of cartilage collagen in different arthritides.

L14 ANSWER 7 OF 9 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1989:25346 BIOSIS

DOCUMENT NUMBER: BA87:13346

TITLE: ACTIVATION OF NEUTRAL METALLOPROTEASE IN HUMAN **OSTEOARTHRITIC KNEE** CARTILAGE EVIDENCE FOR DEGRADATION IN THE CORE PROTEIN OF SULFATED PROTEOGLYCAN.

AUTHOR(S): MARTEL-PELLETIER J; PELLETIER J-P; MALEMUD C J

CORPORATE SOURCE: DEP. MED., WEARN BUILD., ROOM 549, CASE WESTERN RESERVE UNIV., CLEVELAND, OHIO 44106.

SOURCE: ANN RHEUM DIS, (1988) 47 (10), 801-808.
CODEN: ARDIAO. ISSN: 0003-4967.

FILE SEGMENT: BA; OLD
LANGUAGE: English

AB The neutral, metal dependent, proteoglycan degrading **enzymes** (NMPEs) in human **osteoarthritic knee** cartilage homogenates were activated by p-aminophenylmercuric acetate (APMA). The resultant effect on the structure of newly synthesised and already

existing sulphated proteoglycan was measured. Newly synthesised and already existing proteoglycan aggregated to hyaluronic acid was reduced ($p < 0.01$, $p < 0.05$ respectively) when measured by chromatography on Sepharose CL-2B eluted with associative buffer. The APMA activated **enzyme** affected both the newly synthesised and already existing proteoglycan aggregate similarly ($r = 0.79$, $p < 0.001$). **Treatment** of cartilage homogenates with APMA and 1,10-phenanthroline (10 mM) showed that the amount of aggregated proteoglycan was at the control level. The hydrodynamic size of the proteoglycan monomer (A1D1) was also reduced by **treatment** of cartilage homogenates with APMA. Reaggregation experiments with fraction A1D1 and exogenous hyaluronic acid and link protein showed a similar defect in forming proteoglycan aggregates. These data showed that activation of the NMPEs altered the structure of proteoglycan in two ways. The most consistent change was a reduction in the ability of proteoglycan to form aggregates with hyaluronic acid. This was likely to have occurred via a cleavage of the core protein in or around the hyaluronic acid binding globular domain. A second alteration probably includes a limited proteolytic cleavage in the remainder of the core protein.

L14 ANSWER 8 OF 9 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000033966 EMBASE
TITLE: Conservative management of the **osteoarthritic**
knee.
AUTHOR: Troum O.M.; Lemoine C.
CORPORATE SOURCE: Dr. O.M. Troum, School of Medicine, University of Southern California, 2336 Santa Monica Boulevard, Santa Monica, CA 90404, United States
SOURCE: Current Opinion in Orthopaedics, (2000) 11/1 (3-8).
Refs: 41
ISSN: 1041-9918 CODEN: COORE
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 031 Arthritis and Rheumatism
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Osteoarthritis (OA) is the most common type of arthritis affecting synovial joints. Recent advances have altered the traditional progression of medical therapy for OA and have supplied new alternatives for the **treatment** of refractory OA. The new selective cyclooxygenase-2-inhibitory nonsteroidal anti-inflammatory drugs, celecoxib and rofecoxib, have significantly improved safety profiles, particularly with respect to serious gastrointestinal side effects and platelet inhibition. They should be used preferentially in higher-risk patients. Intra-articular viscosupplementation of the knee with exogenous hyaluronic acid has been approved by the US Food and Drug Administration as a medical device for the **treatment** of OA of the knee. It is reportedly as effective as nonsteroidal anti-inflammatory drugs for moderate OA of the knee. Finally, arthroscopic knee-joint lavage, with or without **steroids**, is another alternative for the **treatment** of knee OA; it should be considered before surgery is contemplated. Agents that may prevent cartilage degradation, such as the nutraceuticals (glucosamine sulfate, chondroitin sulfate, and collagen hydrolysate) or inhibitors of nitric oxide or metalloproteinases, may prove beneficial but are still under investigation.

L14 ANSWER 9 OF 9 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 88245347 EMBASE
DOCUMENT NUMBER: 1988245347

TITLE: Activation of neutral metalloprotease in human **osteoarthritic knee** cartilage: Evidence for degradation in the core protein of sulphated proteoglycan.

AUTHOR: Martel-Pelletier J.; Pelletier J.-P.; Malemud Ch. J.

CORPORATE SOURCE: Unite des Maladies Rhumatismales, Research Centre, Hopital Notre-Dame, University of Montreal, Montreal, Que., Canada

SOURCE: Annals of the Rheumatic Diseases, (1988) 47/10 (801-808).
ISSN: 0003-4967 CODEN: ARDIAO

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal

FILE SEGMENT: 031 Arthritis and Rheumatism

LANGUAGE: English

SUMMARY LANGUAGE: English

AB The neutral, metal dependent, proteoglycan degrading **enzymes** (NMPEs) in human **osteoarthritic knee** cartilage homogenates were activated by p-aminophenylmercuric acetate (APMA). The resultant effect on the structure of newly synthesised and already existing sulphated proteoglycan was measured. Newly synthesised and already existing proteoglycan aggregated to hyaluronic acid was reduced ($p < 0.01$, $p < 0.05$ respectively) when measured by chromatography on Sepharose CL-2B eluted with associative buffer. The APMA activated **enzyme** affected both the newly synthesised and already existing proteoglycan aggregate similarly ($r = 0.79$, $p < 0.001$). **Treatment** of cartilage homogenates with APMA and 1,10-phenanthroline (10 mM) showed that the amount of aggregated proteoglycan was at the control level. The hydrodynamic size of the proteoglycan monomer (A1D1) was also reduced by **treatment** of cartilage homogenates with APMA. Reaggregation experiments with fraction A1D1 and exogenous hyaluronic acid and link protein showed a similar defect in forming proteoglycan aggregates. These data showed that activation of the NMPEs altered the structure of proteoglycan in two ways. The most consistent change was a reduction in the ability of proteoglycan to form aggregates with hyaluronic acid. This was likely to have occurred via a cleavage of the core protein in or around the hyaluronic acid binding globular domain. A second alteration probably includes a limited proteolytic cleavage in the remainder of the core protein.

L8 ANSWER 1 OF 8600 MEDLINE
ACCESSION NUMBER: 2002209418 IN-PROCESS
DOCUMENT NUMBER: 21940459 PubMed ID: 11943764
TITLE: The Drosophila homolog of NTF-2, the nuclear transport factor-2, is essential for immune response.
AUTHOR: Bhattacharya Ananya; Steward Ruth
CORPORATE SOURCE: Waksman Institute, Department of Molecular Biology and Biochemistry, Cancer Institute of New Jersey, Rutgers University, 190 Frelinghuysen Road, Piscataway, NJ 08854-8020, USA.
SOURCE: EMBO Rep, (2002 Apr) 3 (4) 378-83.
PUB. COUNTRY: Journal code: 100963049. ISSN: 1469-221X.
England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020412
Last Updated on STN: 20020412
AB Nuclear transport factor-2 (NTF-2) functions in yeast and mammalian cell culture in targeting proteins into the nucleus. The Drosophila homolog, DNTF-2, is an essential component of the nuclear import machinery, since ntf mutants are lethal. Interestingly, hypomorphic alleles show specific phenotypes. Some are viable, but the number of ommatidia in the eye is severely reduced. The immune response in the Drosophila larval fat body is also affected; the three NF-kappaB/Rel proteins Dorsal, Dif and Relish do not target to the nucleus after infection, and, consequently, the expression of the **anti-microbial** peptide genes drosomycin, attacin and drosocin is severely impaired. Hence, in spite of its general requirement in many developmental processes, DNTF-2 has a higher specific requirement in the development of the eye and in the immune response. We also found that DNTF-2 interacts directly with Mbo/DNup88, which does not contain phenylalanine-glycine-rich repeats, but has been shown to function in the import of Rel proteins.

L8 ANSWER 2 OF 8600 MEDLINE
ACCESSION NUMBER: 2002183871 IN-PROCESS
DOCUMENT NUMBER: 21910599 PubMed ID: 11915876
TITLE: Non-trachomatous corneal opacities in the Gambia--aetiology and visual burden.
AUTHOR: Bowman R J C; Faal H; Dolin P; Johnson G J
CORPORATE SOURCE: International Centre for Eye Health London, UK..
richardbowman@iceh.freeserve.co.uk
SOURCE: Eye, (2002 Jan) 16 (1) 27-32.
Journal code: 8703986. ISSN: 0950-222X.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20020403
Last Updated on STN: 20020403
AB AIMS: National blindness surveys conducted in the Gambia in 1986 and 1996 showed an increase in blindness and visual impairment from non-trachomatous opacity. This study aimed to investigate the aetiology of these opacities and to assess the resulting visual burden. METHODS: A population-based, randomised blindness survey was conducted in the Gambia in 1996. Patients with visual impairment or blindness were examined by an ophthalmologist with a slit lamp. Causes of corneal opacity were determined as accurately as possible by clinical history and examination. RESULTS: A total of 154 patients with non trachomatous corneal opacity were examined of whom 39 had bilateral opacities and 115, unilateral. Causes included corneal infection, measles/vitamin A deficiency, harmful traditional practices and trauma (unilateral scarring). Overall, corneal

pathology alone was responsible for bilateral visual impairment or blindness in 19 (12%) patients and unilateral visual impairment or blindness in 88 (57%) patients. Those patients with bilateral visual impairment or blindness (mean age 59, SD) were older (P= 0.003) than others (mean age 44, SD = 20). The use of harmful traditional eye practices was associated with bilateral corneal blindness or visual impairment (RR = 2.63, 95% CI 1.11-6.21, P = 0.04). Although none of the corneal scars reported here were attributed to trachoma, in patients over the age of 45, the prevalence of trachomatous conjunctival scarring in this group was 38.8% compared to 19.4% of the whole nationwide sample. DISCUSSION: Strategies for the prevention (including the quest for cheaper anti-microbial drugs and co-operation with traditional healers) and surgical treatment of these corneal opacities are discussed.

L8 ANSWER 3 OF 8600 MEDLINE

ACCESSION NUMBER: 2002178591 IN-PROCESS

DOCUMENT NUMBER: 21908509 PubMed ID: 11911595

TITLE: Escherichia coli isolates from young calves in Bavaria: in vitro susceptibilities to 14 anti-microbial agents.

AUTHOR: Werckenthin C; Seidl S; Riedl J; Kiossis E; Wolf G; Stolla R; Kaaden O R

CORPORATE SOURCE: Institut fur Medizinische Mikrobiologie, Infektions- und Seuchenmedizin, Ludwig-Maximilians-Universitat Munchen, Munich, Germany.. christiane.werckenthin@micro.vetmed.uni-muenchen.de

SOURCE: J Vet Med B Infect Dis Vet Public Health, (2002 Feb) 49 (1) 61-5.

Journal code: 100955260. ISSN: 0931-1793.

PUB. COUNTRY: Germany: Germany, Federal Republic of Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 20020326

Last Updated on STN: 20020326

AB During the occasional testing of Escherichia coli from faecal samples of young calves we observed multi-resistant isolates. Because of the significance of E. coli as an indicator bacterium for resistance trends we tested E. coli populations of young calves over a longer period. Here we present the results of a retrospective study comparing isolates from 1998 to 2000. Moreover, we compared, in a clinical study, the resistance rates of E. coli populations from 67 hospitalized calves both before and after hospitalization (with or without anti-microbial therapy), and with their anamnestic data of antibiotic usage. The highest resistance rates were found to be more than 80% for tetracyclines, ampicillin, sulfonamide/trimethoprim combinations, and chloramphenicol. A significant increase or decrease over the years was not observed. In analysing the data of hospitalized calves, an increase of resistance to some anti-microbials had to be registered that seemed to be connected with the selective pressure due to agents used in the clinic. In comparing anamnestic data and resistance rates it became obvious that reliable data are not easily available and that a number of potential anti-microbial influence factors have to be taken into account.

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U HAVE REQUESTED DATA FROM FILE 'MEDLINE, EMBASE' - CONTINUE? (Y)/N:yes

L9 ANSWER 1 OF 3 MEDLINE
ACCESSION NUMBER: 1998317003 MEDLINE
DOCUMENT NUMBER: 98317003 PubMed ID: 9653043
TITLE: Microbial corruption of the chemokine system: an expanding paradigm.
AUTHOR: Pease J E; Murphy P M
CORPORATE SOURCE: Department of Applied Pharmacology, Imperial College School of Medicine at the National Heart and Lung Institute, Dovehouse Street, London, SW3 6LY, UK.
SOURCE: SEMINARS IN IMMUNOLOGY, (1998 Jun) 10 (3) 169-78. Ref: 80
Journal code: A61; 9009458. ISSN: 1044-5323.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980820
Last Updated on STN: 19980820
Entered Medline: 19980811

AB The chemokine signaling system includes more than 40 secreted pro-inflammatory peptides and 12 G protein-coupled receptors that together orchestrate specific leukocyte trafficking in the mammalian immune system, ideally for anti- microbial defense and tissue repair processes. Paradoxically and perversely, some chemokines and chemokine receptors are also promicrobial factors and facilitate infectious disease, the result of either exploitation or subversion by specific microbes. Two modes of exploitation are known: usage of cellular chemokine receptors for cell entry by intracellular pathogens, including HIV, and usage of virally-encoded chemokine receptors for host cell proliferation. Likewise, two modes of subversion are known: virally-encoded chemokine antagonists and virally-encoded chemokine scavengers. Understanding how microbes turn the tables on the chemokine system may point to new methods to prevent or treat infection, or, more generally, to treat inappropriate chemokine-mediated inflammation.
Copyright 1998 Academic Press.

L9 ANSWER 2 OF 3 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000435471 EMBASE
TITLE: First line of defense: The role of the intestinal epithelium as an active component of the mucosal immune system.
AUTHOR: Pitman R.S.; Blumberg R.S.
CORPORATE SOURCE: R.S. Pitman, Gastroenterology Division, Brigham and Women's Hospital, Harvard Medical School, 75 Francis Street, Boston, MA 02115, United States
SOURCE: Journal of Gastroenterology, (2000) 35/11 (805-814).
Refs: 121
ISSN: 0944-1174 CODEN: JOGAET
COUNTRY: Japan
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 026 Immunology, Serology and Transplantation
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Over the past decade, many studies have revealed the immunological importance of IECs, not only in maintaining a physical barrier to the

external environment but also by functioning alongside cells of the immune system to prevent infection and epithelial injury (summarized in Fig. 1). Intestinal epithelial cells secrete a variety of extrinsic factors, ranging from those which facilitate repair of damaged tissue, such as ITF, to mucin and **anti-microbial** peptides which directly inhibit bacterial growth across the epithelial monolayer. In addition to those mechanisms which are reliant upon the inherent properties of the epithelium, IECs also function by directly influencing local immune responses. Through the expression of adhesion molecules, costimulatory factors, and a vast array of cytokines, epithelial cells can affect such processes as leukocyte infiltration and IEL growth, development, and responsiveness to antigenic stimuli. The intestinal epithelia may also play a role in processing and presenting luminal antigens to adjacent lymphocyte populations, thereby directing immune responses to specific foreign agents to which the monolayer is exposed. The combination of epithelial cell properties so far described implicates IECs as cells crucial in maintaining the intestinal mucosa in a constant state of immune responsiveness. IECs can be thus defined as essential components of the mucosal immune system.

L9 ANSWER 3 OF 3 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1998251867 EMBASE
TITLE: Microbial corruption of the chemokine system: An expanding paradigm.
AUTHOR: Pease J.E.; Murphy P.M.
CORPORATE SOURCE: J.E. Pease, Department of Applied Pharmacology, Imperial College School of Medicine, National Heart and Lung Institute, Dovehouse Street, London SW3 6LY, United Kingdom
SOURCE: Seminars in Immunology, (1998) 10/3 (169-178).
Refs: 80
ISSN: 1044-5323 CODEN: SEIME2
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The chemokine signaling system includes more than 40 secreted pro-inflammatory peptides and 12 G protein-coupled receptors that together orchestrate specific leukocyte trafficking in the mammalian immune system, ideally for **anti-microbial** defense and **tissue repair** processes. Paradoxically and perversely, some chemokines and chemokine receptors are also promicrobial factors and facilitate infectious disease, the result of either exploitation or subversion by specific microbes. Two modes of exploitation are known: usage of cellular chemokine receptors for cell entry by intracellular pathogens, including HIV, and usage of virally-encoded chemokine receptors for host cell proliferation. Likewise, two modes of subversion are known: virally-encoded chemokine antagonists and virally-encoded chemokine scavengers. Understanding how microbes turn the tables on the chemokine system may point to new methods to prevent or treat infection, or, more generally, to treat inappropriate chemokine-mediated inflammation.

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L1 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:678969 CAPLUS
DOCUMENT NUMBER: 133:221689
TITLE: A *Bacillus* sp. which produce
polyhydroxyalkanates
INVENTOR(S): Lee, Young-Ha; Oh, Suk-Hun; Hong, Sung-Joo
PATENT ASSIGNEE(S): Hanhwa Chemical Co., Ltd., S. Korea
SOURCE: Repub. Korea, No pp. given
CODEN: KRXXFC
DOCUMENT TYPE: Patent
LANGUAGE: Korean
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
KR 129117	B1	19980404	KR 1994-14344	19940622
AB	Bacillus sp. KCTC8552P2 (<i>Bacillus thuringensis</i> H0079) capable of biosynthesizing polyhydroxybutyrate(PHB)-polyhydroxyvalerate(PHV) copolymer including about 90 mol% of 3-hydroxyvalerate(HV) unit is claimed. PHB-PHV copolymer is synthesized in the medium containing glucose as main substrate and propionate as cosubstrate by the single step batch culture. When the concn. of propionate in the medium become 1.0%, max. yield of dry cell wt. and polyhydroxyalkanate biomass indicate 7.3 g/l and 43.9% resp. Copolymer obtained is used for the material of biothermoplastics.			

L1 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:478320 CAPLUS
DOCUMENT NUMBER: 115:78320
TITLE: The degradation of shampoo bottles in a lake
AUTHOR(S): Brandl, Helmut; Puechner, Petra
CORPORATE SOURCE: Inst. Pflanzenbiol., Univ. Zurich, Zurich, 8008, Switz.
SOURCE: NATO ASI Ser., Ser. E (1990), 186(Novel Biodegrad. Microb. Polym.), 421-2
CODEN: NAESDI
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Expts. were carried out in Lake Lugano, Switzerland, to study the biodegradn. of poly(.beta.-hydroxyalkanate) (PHA) in an aquatic ecosystem under natural conditions. Com. available plastic articles made from PHA, such as bottles and films, were incubated for 250 days in a water depth of 85 m. Shampoo bottles were positioned precisely on the sediment surface using a small manned submarine. An expected life span of 10 yr for this specific bottle type was calcd. The results demonstrate that in an aquatic ecosystem even under extreme conditions (low temps., high pressure, no sunlight) plastic articles made from PHA are degraded.

L1 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1991:20781 CAPLUS
DOCUMENT NUMBER: 114:20781
TITLE: Accumulation of a polyhydroxyalkanoate-containing primarily 3-hydroxydecanoate from simple carbohydrate substrates by *Pseudomonas* sp. strain NCIMB 40135
AUTHOR(S): Haywood, Geoffrey W.; Anderson, Alistair J.; Ewing, David F.; Dawes, Edwin A.
CORPORATE SOURCE: Dep. Appl. Biol., Univ. Hull, Hull, HU6 7RX, UK
SOURCE: Appl. Environ. Microbiol. (1990), 56(11), 3354-9
CODEN: AEMIDF; ISSN: 0099-2240
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A no. of *Pseudomonas* species have been identified which accumulate a polyhydroxyalkanoate contg. mainly 3-hydroxydecanoate monomers from sodium gluconate as the sole carbon source. One of these, *Pseudomonas* sp. strain NCIMB 40135, was further investigated and shown to accumulate such a polyhydroxyalkanoate from a wide range of carbon sources (C2-C6); however, when supplied with octanoic acid it produced a polyhydroxyalkanoate contg. mainly 3-hydroxyoctanoate monomers. Polymer synthesis occurred in batch culture after cessation of growth due to exhaustion of nitrogen. In continuous culture under nitrogen limitation, up to 16.9% polyhydroxyalkanoate was synthesized from glucose as the carbon source. The monomer units are mainly of the R-(-) configuration. NMR studies confirmed the compn. of the polymer. Differential scanning calorimetry suggested that the solvent-extd. polymer contained a significant proportion of cryst. material. The wt.-av. mol. wt. of the polymer from glucose-grown cells was 143,000.

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